and 46 asymptomatic patients with a major cardiovascular risk factor (28 with familial hypercholesterolemia and 18 with untreated essential hypertension; age 14–70 years). 2-D, M-mode and Doppler echocardiography were performed to assess LV isovolumic relaxation time (IRT), peak velocity of early (E) and late (A) filling, E/A ratio, E deceleration time (DT), and LV mass (LVMI). PWV was measured by two pressure transducers placed over the right carotid and femoral pulses. Central pressure waveforms were recorded by high fidelity carotid applanation tonometry to obtain augmentation index (AI) and pressure wave transit time (TT = time interval from foot of carotid waveform to inflection point; two-way travel time from the recording site to effective reflection site). In the whole population, we found the following relationships:

| | IRT | E | A | E/A | DT |
|---|---|---|---|---|---|
| Age | 0.52§ | −0.50§ | 0.71§ | −0.74§ | 0.22 |
| SBP | 0.46§ | −0.24* | 0.49§ | −0.44§ | 0.29** |
| PWV | 0.45§ | −0.49§ | 0.65§ | −0.68§ | 0.34** |
| AI | 0.50§ | −0.30** | 0.48§ | −0.49§ | 0.12 |
| TT | −0.48§ | 0.30** | −0.53§ | 0.54§ | −0.11 |
| LVM | 0.39** | −0.44§ | 0.39** | −0.46§ | 0.09 |

SBP = systolic blood pressure; *$p < 0.05$; **$p < 0.01$; §$p < 0.001$

Relations of age and SBP with LV diastolic indices became weak or lost statistical significance after controlling for PWV, AI and TT. *Conclusion:* Higher PWV, stronger and earlier reflected pressure waves waves are related to prolonged LV relaxation and deceleration time, slower rapid filling and enhanced atrial contraction. These data suggest that arterial stiffening and consequent early pressure wave reflection impairs LV diastolic function and contributes to diastolic abnormalities commonly associated with aging, hypertension and atherosclerosis.

## 1053-18 Altered Plaque Distribution in Hypertensive Patients

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Hypertension has been associated with abnormal endothelial function and accelerated atherosclerosis but in vivo coronary plaque morphology and distribution in hypertensive (HTN) patients (pts) is not known. We therefore examined the distribution of atherosclerotic plaque at a stenotic site with intravascular ultrasound, prior to any intervention, in 38 HTN and 14 non-HTN pts. Luminal, arterial and plaque area were measured. Plaque thickness was measured along 180 diameters along the line through the luminal centroid and plaque eccentricity index was calculated as the ratio of the smallest to largest plaque diameter. Plaque morphology was assessed by measuring the arc of calcium and measuring non-calcified plaque density using video densitometry. *Results:* Fifty two patients (37 men, 15 women, age 57.8 ± 10.1 years) underwent IVUS imaging of the stenotic coronary artery (16 RCA, 28 LAD, 8 CX). HTN pts had similar plaque and luminal areas ($p$ = ns) as compared to controls but a larger percent cross-sectional plaque burden (76.4 ± 10.3% vs 67.6 ± 16.6%, $p < 0.05$). Plaque morphology was the same in HTN and non-HTN pts. There was, however, a higher eccentricity index in HTN pts (0.35 ± 0.20 vs 0.19 ± 0.23, $p < 0.05$) as compared to normotensives indicating a more concentric distribution of plaque. *Conclusion:* Pts with HTN have a different plaque distribution at the stenotic site as compared to non-HTN pts; the HTN plaque occupies more of the cross-sectional arterial area and is more concentric. Future prospective investigation with sequential IVUS exams should explore the mechanism of altered plaque distribution in HTN pts and the implications for coronary intervention.

# 1054 Risk Factors in Cardiovascular Disease

Wednesday, March 19, 1997, 9:00 a.m.–11:00 a.m.
Anaheim Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

## 1054-7 Appraising Risk of Cardiovascular Disease Among Treated Hypertensives

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Risk functions that ignore pharmacologic treatment may inaccurately estimate both disease risk and the benefits of risk factor interventions. We developed cardiovascular disease (CVD) and coronary heart disease (CHD) risk functions for treated hypertensives aged 40–79 years (Treated; n = 7,955 person-exams) via logistic regression using data from the Framingham Heart Study and the Framingham Offspring Study. Age, sex, blood pressure (DBP or SBP), diabetes, current smoking, and cholesterol were included in the models. We compared risk factor coefficients in the Treated models to those in models for all other subjects (Others; viz., normotensives and untreated hypertensives; n = 41,907 person-exams). Next, we estimated the change in odds of CVD and CHD due to reductions in DBP using the Treated functions and compared them to estimates using models that combined Treated with Others (Combined). The coefficients for age, blood pressure (DBP or SBP), diabetes and cholesterol differed significantly (all $p < 0.05$) between the models for Treated and Others indicating differences in the predictive power of these risk factors. The Combined models underestimated risks below 90 mm Hg DBP and overestimated risks thereafter. Estimates of percent change in the odds of CVD and CHD, respectively, due to a 10 mm Hg reduction in DBP were 15% and 16% (Treated models) compared to 24% and 22% (Combined models). The Treated risk functions more accurately predicted CVD and CHD events and estimates of disease prevention due to reductions in DBP were consistent with the results of clinical trials of anti-hypertensive drugs.

## 1054-8 Fasting Bloodglucose Level Is an Independent Predictor of Cardiovascular Mortality in Healthy Norwegian Men. Results From 22 Years Follow Up

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Fasting bloodglucose level is important in detecting Diabetes mellitus, but the impact of mildly elevated levels within the normal range in healthy persons, has not been explored.

Fasting bloodglucose was measured during a cardiovascular screening survey in 2014 healthy men, aged 40–59, in 1972–75. Complete 22 years mortality data were classified according to ICD 9 standards. Total mortality was 23.8% (n = 480), of which cardiovascular (CV) death accounted for 53.8%. The 12 hours fasting blood glucose levels were ranged between 2.9 to 8.0 mmol/l, 99% were under 6.0 mmol/l. The table gives cardiovascular and noncardiovascular mortality in quartiles of fasting blood glucose.

| Fasting blood glucose: | CV mortality: | Non-CV mortality: |
|---|---|---|
| ≤ 4.0 mmol/l (n = 575) | 12.7% | 11.1% |
| 4.1–4.3 mmol/l (n = 462) | 11.0% | 10.8% |
| 4.4–4.6 mmol/l (n = 488) | 10.4% | 11.1% |
| ≥ 4.7 mmol/l (n = 484) | 17.1% | 10.1% |

In the 4'th quartile a threshold can be detected at 4.9 mmol/l, CV mortality under the threshold 11.8% and over 19.5%. According to Cox regression analysis bloodglucose level over 4.9 mmol/l was related to increased CV mortality (Relative risk = 1.5; 95% CI: 1.1–1.8) after accounting for age, smoking habits, serum lipids, resting heartrate, bloodpressure, body mass index, FEV'1 and physical fitness.

In conclusion, fasting bloodglucose over 4.9 mmol/l is an independent risk factor which identifies middle-aged non-diabetic healthy men at increased risk for cardiovascular death.

## 1054-9 Unrecognized Exertional Heat Illness as a Risk Factor for Exercise-Related Sudden Cardiac Death Among Young Adults

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Exercise-related death (ERD) among recruits usually occurs during middle distance running in the first weeks of training in the hot season, early in the day when the wet bulb globe temperature index (WBGT) is considered safe. Exertional heat illness (EHI), which is largely preventable, occurs under similar circumstances. We showed that risk of EHI increases ≥ 15-fold when the prior day's WBGT was ≥ 75ºF. To test the hypothesis that unrecognized EHI might contribute to sudden cardiac ERD, both explained and unexplained by preexisting heart disease, we determined etiology for all 94 ERDs and 12 sudden cardiac deaths at rest among 5.5 million recruits in US military basic training for 1977–1990. Full autopsy and toxicology reports were obtained for 101/106 deaths and eyewitness accounts and clinical records for > 75% of cases. Control cases unrelated to heat were deaths at rest and 6 non-cardiac explained ERDs. The frequency of significant heat stress, defined as a prior day WBGT ≥ 75ºF, was 2/18 Controls, 15/39 explained cardiac ERD, 8/15